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**TESTIMONY ON 29 CFR PARTS 1910, 1915, 1926,
AND 1928
INDOOR AIR QUALITY; PROPOSED RULE**

**DEPARTMENT OF LABOR, OCCUPATIONAL SAFETY AND
HEALTH ADMINISTRATION**

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INTRODUCTION

My name is Jonathan M. Samet. I am a medical doctor, trained in Internal Medicine and the subspecialty of Pulmonary Medicine. I received an A.B. degree from Harvard College and an M.D. degree from the University of Rochester School of Medicine and Dentistry. I am also an epidemiologist and have received a Master of Science in epidemiology from the Harvard School of Public Health. Most of my professional career has been spent at the University of New Mexico School of Medicine, where I most recently had the title of Professor of Medicine and Chief of the Pulmonary and Critical Care Division of the Department of Medicine. I recently assumed my present position as Professor and Chair of the Department of Epidemiology of the Johns Hopkins University School of Hygiene and Public Health.

My clinical practice has covered the full range of pulmonary diseases but more recently it has increasingly focused on the diagnosis and management of patients with occupational and environmental lung diseases. I have provided clinical care for patients with problems stemming from indoor air pollution exposures and for patients with the clinical syndromes referred to as Sick Building Syndrome and Multiple Chemical Sensitivity. In New Mexico, my research emphasized the effects of inhaled agents on health and particularly outdoor and indoor air pollutants, including radon, nitrogen dioxide (NO₂), and environmental tobacco smoke (ETS). This research has addressed the non-malignant and malignant effects of ETS, nitrogen dioxide and respiratory illnesses, and radon and lung cancer. I have also conducted studies directed at time-activity patterns and personal exposures to pollutants and I have studied the effects of active smoking. I have authored or co-authored many scientific papers on these topics and with a colleague at the Harvard School of Public Health, John D. Spengler, Ph.D., I edited a book, *Indoor Air Pollution: A Health Perspective*, which was published by Johns Hopkins University Press in 1991. More recently, I edited *Epidemiology of Lung Cancer* which was published by Marcel Dekker, Inc. in 1994.

I have served on a number of committees and advisory groups concerned directly or indirectly with indoor air quality and health. These include the Indoor Air Quality and Total Human Exposure Committee of the Science Advisory Board of the Environmental Protection Agency; the National Air Conservation Commission of the American Lung Association; and SSPC 62 of the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE)

which is charged with revising the organization's Standard 62. I also served on the Working Group on Tobacco Smoking of the International Agency for Research on Cancer and was a Consulting Scientific Editor for the 1986 Report of the Surgeon General on involuntary smoking. I was subsequently the Senior Scientific Editor for the 1990 Report of the Surgeon General on smoking cessation. I am presently Chairman of the Biological Effects of Ionizing Radiation (BEIR) VI Committee of the National Research Council. I served on BEIR IV and was Chairman of the Panel on Dosimetric Assumptions Affecting the Application of BEIR IV Risk Estimates.

THE PROBLEM OF INDOOR AIR POLLUTION

Indoor air pollution is a complex societal problem. We spend most of our time indoors and we expect that our indoor environments will not be a cause of discomfort and disease. Mounting evidence, however, has shown that indoor air pollution can cause a wide range of adverse effects, ranging from discomfort and annoyance at the least severe to death at the most severe. As we have characterized time-activity patterns of the population, that is the locations where people spend time and the amounts of time spent in these locations, we have gained an understanding of the contributions of various indoor environments to personal exposures to pollutants. Studies of time-activity patterns and personal exposures indicate dominant contributions from indoor environments for exposures to many pollutants that have adverse consequences. As would be anticipated, the workplace and the home are the strongest contributors for most agents.

The Occupational Safety and Health Administration (OSHA) is addressing the adverse effects of exposures received in the workplace in its Proposed Rules. This testimony, presented in support of the Proposed Rules, covers the adverse health effects of indoor air pollution and the mechanisms underlying these effects and considers the potential benefits of compliance with the rules proposed by OSHA.

The Proposed Rules are timely. Data on the health effects of indoor air pollution have largely been collected over the last 20 years; the evidence is now sufficient to identify some adverse effects with certainty and the experience of the diverse professionals concerned with indoor air pollution, documented in the submissions in response to OSHA's Request for Information (56 FR 47892), suggests that adverse effects may be increasing. Additionally, a

large number of professionals, including engineers, industrial hygienists, and others now work to solve problems of indoor air quality in non-industrial buildings. Unfortunately, because of the proprietary nature of this work, there is no quantitative information available on the numbers of buildings which have had problems of severe enough magnitude to require evaluation and remediation. Thus, even though there are acknowledged uncertainties in our understanding of indoor air pollution and health, the public health burden posed by the adverse consequences of indoor air pollutants has become sufficient to warrant implementation of regulations by OSHA.

However, the Proposed Rules address a problem quite different from the regulation of a single agent that may be linked to one or more specific health outcomes, e.g., asbestos and asbestosis, mesothelioma, and lung cancer. Indoor air may be contaminated by many different pollutants causing disease through diverse mechanisms. The pollutants exist in complex mixtures and the presence of one pollutant in the mixture may augment (synergism) or diminish (antagonism) the effect of another pollutant. Environmental tobacco smoke, a pollutant considered in the Proposed Rules, is itself a complex mixture of gaseous and particulate agents produced by tobacco combustion. Some of the approaches outlined in the Proposed Rules are appropriately not directed at single pollutants but at managing the problem of indoor air pollution at a more holistic level. This type of approach is warranted rather than the presently unworkable alternative of proposing concentration guidelines for individual pollutants or for pollutants that might be considered as indicators for complex mixtures.

Multiple mechanisms underlie the adverse effects of indoor air pollution. The principal mechanisms include immediate and delayed hypersensitivity, infection, irritation of mucous membrane receptors, inflammation of epithelial and alveolar surfaces, interference with oxygen transport, and carcinogenesis, and some effects probably have a neurophysiological basis. For some pollutants and adverse effects, underlying pathophysiologic mechanisms have been advanced but remain to be established. For example, the Sick Building Syndrome has been postulated to reflect irritation of receptors in mucous membranes by volatile organic compounds (Molhave 1992). Some effects of indoor air pollution may reflect several different mechanisms. Thus, Sick Building Syndrome has also been linked to biological agents in addition to volatile organic compounds (Institute of Medicine, 1993).

While these mechanisms are presently considered to be the basis for most of the adverse consequences of indoor air pollution, the specific causal pathways remain to be established for a number of the outcomes. Sick Building Syndrome, for example, has been associated with inadequately maintained heating, ventilating, and air-conditioning systems, although causal links to specific agents have not uniformly been made in investigations of individual buildings (Marbury and Woods 1991).

The spectrum of the adverse outcomes also differs from the clinically defined effects of most regulated occupational agents; For indoor air pollution, some adverse effects are well characterized and represent distinct clinical entities, e.g., hypersensitivity pneumonitis and pneumonia caused by *Legionella*. However, some of the most frequent adverse consequences, discomfort, irritation, and symptoms compatible with the Sick-Building Syndrome, are not yet well characterized from a clinical perspective, in spite of their prevalence in the workforce, which is likely to be high, and their significant impact on productivity. Even though these symptom responses might not be classified as diseases, they adversely affect health when defined broadly, as by the World Health Organization, to include well-being. The Proposed Rules acknowledge this range of responses and the complexities of defining some adverse consequences of indoor air pollution without ambiguity should not dissuade OSHA from proceeding.

Table 1 provides a classification of the full range of responses to indoor air pollution, including categories for disease, impairment, symptoms, increased risk, and perceptions (Samet 1994). This classification is based on a plenary presentation made at Indoor Air '93, an international congress on indoor air that is held every three years. Each category in this classification is treated below and examples provided. The classification serves to illustrate the range of responses of concern with regard to indoor air pollution and the extent of the adverse effects that OSHA needs to address.

Few quantitative estimates of the burden of disease posed by each of these categories have been made. Risk assessment not only requires the determination that an agent poses a hazard but also characterization of the relationship between dose and response and of the distribution of exposure. The requisite data are not available for most indoor air contaminants of concern in the work place, although risk estimates have been made for ETS and asbestos.

If appropriately catalogued, the existing data might prove more informative with regard to exposures to some indoor air pollutants and various administrative data bases assembled for

health care could provide insights into the frequency of some key conditions and illnesses, such as pneumonia caused by *Legionella* and hypersensitivity pneumonitis. The prevalence of symptoms and discomfort could be determined by survey techniques. Creating a registry of buildings where complaints compatible with Sick Building Syndrome have been investigated would also be informative. We lack information on dose-response relationships for a number of key pollutants. Only careful epidemiological and toxicological research can address this gap.

Clinically Evident Disease: While exposures to indoor air pollutants are universal, clinically-diagnosed cases of pollution-related disease appear to be relatively infrequent, although most clinicians do not actively pursue associations between disease and environmental exposures, including indoor air pollution. In fact, a clear relationship with indoor pollution may be clinically very difficult to establish, because even the patient may be unaware of the relevant exposures. In the case of such a clinically evident disease, a link can be established to an indoor pollutant by specific diagnostic tests (Table 2). For example, an appropriate clinical picture and an elevated serum precipitin titer are sufficient to document hypersensitivity pneumonitis due to thermophilic actinomycetes contaminating an air conditioning system (Weissman and Schuyler 1991). The level of carbon monoxide bound to hemoglobin (carboxyhemoglobin) provides a marker of exposure to concentrations of carbon monoxide associated with carbon monoxide poisoning. Skin tests and serologic tests can provide evidence of sensitization to antigens that produce disease through immediate hypersensitivity responses.

In classifying illnesses associated with public and commercial building environments, this category of adverse effects, e.g., hypersensitivity pneumonitis, has been referred to as specific building-related illnesses or building-related illness, as in the OSHA Proposed Rules (American Thoracic Society 1990; Marbury and Woods 1991). However, the distinction between specific building-related illnesses and the non-specific syndrome referred to as Sick (or Tight) Building Syndrome rests on the establishment of a clinical diagnosis for the former category. This group of adverse effects, clinically evident disease, should be recognized as unified on this basis.

Lung cancer caused by indoor carcinogens can also be placed in this category, although it is separately considered below under the category, "increased risk of disease". Respiratory carcinogens in

indoor air include ETS, radon, and asbestos. In the workplace, ETS is of principal concern. The U.S. Environmental Protection Agency has recently made estimates of the number of lung cancer cases attributable to ETS, but did not specifically estimate the number of cases attributable to involuntary exposure in the workplace (U.S. Environmental Protection Agency 1992). Estimates have been made for various scenarios of indoor exposure by the Review Panel of the Health Effects Institute-Asbestos Research (Health Effects Institute Asbestos Research Literature Review Panel 1991).

Exacerbation of Established Disease: Conditions that may be exacerbated by indoor air pollution are common in the work force. As much as 30 to 40 percent of the population is atopic, that is allergic, and at risk for hypersensitivity responses to indoor allergens (Institute of Medicine, 1993). Asthma, a chronic respiratory disease characterized by hyperresponsiveness of the lung's airways to environmental factors, affects approximately 5% of adults (National Asthma Education Program 1991). Chronic obstructive pulmonary disease or COPD, also a chronic respiratory disease but characterized by permanent reduction of lung function, affects several percent of adults (U.S. Department of Health and Human Services 1984). Coronary heart disease becomes manifest in middle-aged and older adults, also affecting several percent of adults (U.S. Department of Health and Human Services 1990).

These susceptible members of the work force are at greater risk from a variety of indoor pollutants. Indoor exposures to animal danders, molds, and allergens from house dust mites and other insects may both cause and worsen the clinical status of persons with asthma. To date, the evidence on indoor air pollution and asthma primarily comes from research in the indoor environment. However, the same exposures should have comparable effects in other settings, including the workplace. Environmental tobacco smoke may increase the non-specific responsiveness of the lung to environmental stimuli and even trigger attacks of asthma (Samet, Cain et al. 1991). In managing patients with more severe asthma, particularly if therapy has not been effective, my clinical approach includes a detailed assessment of environmental exposures at home and at work. ETS and other inhaled irritants would be anticipated to affect persons with COPD and individuals with coronary heart diseases may be adversely impacted by carbon monoxide.

Increased Risk for Disease: Many pollutants in indoor air are associated with increased risk for a variety of malignant and non-

malignant diseases (Table 3). The evidence supporting the relationships between exposures to these agents and increased risk comes from epidemiological studies, short-term exposures of volunteer subjects, animal studies, and *in vitro* toxicological studies. The population burden of disease attributable to such agents is often estimated using quantitative risk assessment, as has been done for ETS, radon, and asbestos.

Physiologic Impairment: Exposures to indoor pollutants can impair physiological functioning, although not to a degree necessarily associated with disability or disease. For example, exposure to ETS during childhood reduces the rate of lung growth and the maximum level of lung function achieved; the average estimated effect is not anticipated to be clinically detectable nor to be associated with reduced functional capacity (Samet, Cain et al. 1991). Similarly, low levels of carbon monoxide exposure transiently impair oxygen delivery to tissues; however, the impact on exercise capacity is limited and likely to be manifest only during maximal activity (Coultas and Lambert 1991). On the other hand, reduced oxygen transport in the carbon-monoxide-exposed individual with coronary artery disease may increase the likelihood of clinically significant myocardial ischemia. The public health relevance of this category of adverse effects of indoor air pollution has received little consideration to date.

Symptom Responses: Epidemiological evidence links specific indoor air pollutants to a variety of symptoms. Environmental tobacco smoke exposure, for example, has been causally associated with increased risk of respiratory symptoms in children (U.S. Department of Health and Human Services 1986), and some studies indicate increased risk for exposed adults as well. The Sick-Building Syndrome is a non-specific constellation of symptoms characteristically affecting multiple occupants of a building (American Thoracic Society 1990). However, it is very difficult for a clinician to establish an association between symptoms and air pollution exposure in an individual patient and the diagnosis of Sick Building Syndrome should be made in an epidemiologic context, that is with evidence that multiple individuals in the work place have been affected.

Estimates of the burden of symptoms associated with indoor air pollutants have not been made; however, this burden is likely to be substantial because of the high prevalence rates of exposure to agents associated with symptoms. Surveys of the prevalence of

work-related symptoms, although not conducted in random samples of buildings, indicate high prevalence rates for symptoms. Burge and coworkers (Burge, Hedge et al. 1987) described symptom rates in 4,373 office workers in 42 different buildings. Symptoms were considered work related if they occurred more than twice during the previous 12 months and improved on days away from the office. Using this definition, the mean number of work-related symptoms varied across the sample of buildings from approximately 1.5 to 5. Symptoms of eye and upper airway irritation and headaches were common (Table 4).

Perception of Unacceptable Indoor Air Quality: The perception that indoor air quality is unacceptable should be considered as distinct from the symptoms caused by indoor air pollutants. There appears to be a wide range of tolerance of indoor air pollution in the population. For some, unacceptable indoor air quality reduces well-being and for such persons, the perception of indoor air quality as unacceptable should be classified as an adverse health effect in the context of current concepts of health. Judgments as to the acceptability of indoor air quality presumably integrate multiple characteristics of the air, including the presence of odor and irritants, humidity, air movement, and temperature (Berglund and Lindvall 1990; Spengler and Samet 1991). Undoubtedly, there is a range of responses and expectations across the population. Physical and psychological aspects of the environment not directly related to indoor air quality may also influence judgments as to the acceptability of indoor air quality.

The findings of a nationwide survey of U.S. office workers suggest that dissatisfaction with the air quality in offices is common (Woods, Drewry et al. 1987). Of 600 workers surveyed by telephone in 1984, 20% perceived that their work performance was affected "often" or "sometimes". Aspects of indoor air quality that were found to be "very serious" or "serious" by at least 50% of the affected respondents included lack of air movement (67%), being too hot in summer (61%), stagnant or still air (55%), cigarette smoke (54%), being too cold in winter (53%), and being too humid in summer (50%).

Perception of Exposure to Indoor Air Pollutants: The perception of exposure to indoor pollutants should also be regarded as an adverse health effect, if this perception reduces well-being. The range of responses to the perception of exposure is broad, extending from annoyance because of an odor to the sometimes disabling symptom

complex now frequently referred to as "multiple chemical sensitivity". The pathogenetic mechanisms underlying multiple chemical sensitivity remain unknown and may be multiple. The numbers of persons who are adversely affected by the perception of exposure cannot be presently estimated.

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke is among the single pollutants targeted specifically for control in the Proposed Rules. This term refers to the mixture of sidestream smoke and exhaled mainstream smoke that contaminates air in spaces where smoking is taking place. Although referred to as though it were a single agent, ETS is a complex mixture of particles and gases that is known to have many of the same toxic and carcinogenic components that are present in the mainstream smoke inhaled by the active smoker (U.S. Department of Health and Human Services 1986; U.S. Environmental Protection Agency 1992). Markers of tobacco smoke can be measured in smoking-contaminated air, e.g., nicotine, and uptake of ETS components by nonsmokers has been shown using cotinine and other biomarkers.

Active smoking has diverse adverse effects including being a cause of cancer and a number of non-malignant conditions as well. This evidence has been comprehensively reviewed in the Reports of the Surgeon General on smoking and health. In regard to exposures of adult nonsmokers in the workplace to ETS, the literature on active smoking indicates a basis for concern about risk for lung cancer and for heart disease. Active cigarette smoking has long been established as a cause of lung cancer, with the risk varying with the number of cigarettes smoked per day and the duration of smoking, as well as other aspects of smoking behavior and the type of product smoked (U.S. Department of Health and Human Services 1982; U.S. Department of Health and Human Services 1989). The risk for coronary heart disease is also increased by cigarette smoking with the risk depending most directly on being a current smoker and the number of cigarettes smoked per day (U.S. Department of Health and Human Services 1983; U.S. Department of Health and Human Services 1990).

In fact, a substantial epidemiologic literature indicates increased risk for lung cancer in never smokers exposed to ETS (U.S. Environmental Protection Agency 1992). These epidemiologic studies are largely of the case-control design, comparing exposures of never smokers with lung cancer to those of control never smokers without lung cancer. The principal exposure variable assessed in these studies has been marriage to a smoker, based on the assumption that never smokers married to smokers sustain greater exposure at home than never smokers married to never smokers. The weight of the evidence indicates increased lung cancer risk for

never smokers married to smokers; the causal nature of this association is further supported by present understanding of respiratory carcinogenesis (U.S. Department of Health and Human Services 1986; U.S. Environmental Protection Agency 1992). Only a few studies have addressed exposure to ETS in the workplace and a precise estimate of the risk of workplace exposure is not available. It would be anticipated that the risk would be variable among workplaces, depending on the level of contamination and the duration of exposure. There is no reason to assume that an agent, tobacco smoke, that has been shown to be a carcinogen when actively inhaled during active smoking and passively inhaled at home, would not be a carcinogen when involuntarily inhaled in the workplace.

For coronary heart disease, the epidemiologic evidence on passive smoking is less abundant, but does indicate increased risk (American Heart Association 1992). Mechanisms have been postulated on the basis by which ETS could cause coronary heart disease and the American Heart Association has concluded that ETS is a cause of coronary heart disease in adults. The workplace has not received specific investigation.

Other effects of ETS exposure have been identified and considered to have a causal relationship to exposure (National Research Council and Committee on Passive Smoking 1986; U.S. Department of Health and Human Services 1986; U.S. Environmental Protection Agency 1993). Children exposed to ETS are at increased risk for lower respiratory illnesses during the first years of life. Additionally, they have increased rates of respiratory symptoms and the lung function of exposed children increases at a lesser rate than for unexposed children. The status of children with asthma is adversely affected by ETS (U.S. Environmental Protection Agency 1992) and ETS is a suspect cause of asthma. ETS exposure could plausibly exacerbate asthma in adults as well. Some studies have indicated that exposure of adults to ETS may also adversely lung function and produce respiratory symptoms, although the evidence has not been judged conclusive (Samet, Cain et al. 1991).

THE POTENTIAL BENEFITS OF THE PROPOSED RULES

The Proposed Rules have two elements, a broad strategy oriented towards achieving acceptable indoor air quality through a compliance program designed to assure indoor air quality through building operation and control approaches directed at individual pollutants or groups of pollutants including ETS. These Proposed

Rules are offered at a time when there are evident gaps in our understanding of indoor air quality and health. Nevertheless, the Proposed Rules offer approaches that build on both the scientific evidence on indoor air pollution and health and on the field experience of persons who operate buildings and evaluate buildings with problems. Benefits for health of the workforce can reasonably be anticipated even though any quantitative estimates of the burden of morbidity and mortality caused by indoor air pollution in the work place are subject to diverse uncertainties. For ETS, exposures can be completely avoided by prohibiting smoking and the alternative strategy of separate smoking areas under negative pressure should minimize exposure of nonsmokers to ETS. OSHA can reasonably project avoidance of the burden of morbidity and mortality associated with ETS with implementation of the Proposed Rules.

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Table 1 A classification of the adverse effects of indoor air pollution.

Clinically evident diseases: Disease for which the usual methods of clinical evaluation can establish a causal link to an indoor air pollutant.

Exacerbation of disease: The clinical status of already established disease is exacerbated by indoor air pollution.

Increased risk for disease: Diseases for which epidemiological or other evidence establishes increased risk in exposed individuals. However, the usual clinical methods indicative of injury typically cannot establish the causal link in an individual patient.

Physiological impairment: Transient or persistent effects on a measure of physiological functioning which are of insufficient magnitude to cause clinical disease.

Symptom responses: Subjectively reported responses which can be linked to indoor pollutants or are attributed to indoor pollutants.

Perception of unacceptable indoor air quality: Sensing of indoor air quality as uncomfortable to an unacceptable degree.

Perception of exposure to indoor air pollutants: Awareness of exposure to one or more pollutants with an unacceptable level of concern about exposure.

Table 2 Selected examples of clinically evident disease linked to indoor air pollution.

Carbon monoxide poisoning

Hemorrhagic pneumonitis from high level of NO₂

Hypersensitivity pneumonitis and humidifier fever

Legionella pneumonia

Cat- and mite-induced asthma

**Table 3 Selected examples of exposure-disease associations
for indoor air pollutants**

Radon: Lung Cancer

**Environmental Tobacco Smoke: Lung cancer, increased lower
respiratory illness in infants**

Benzene: Leukemia

Asbestos: Lung cancer and mesothelioma

Formaldehyde: Nasal cancer

Table 4 Prevalence of selected symptoms in office workers by type of ventilation system*

| Ventilation Type | Symptoms | | | |
|----------------------------|----------|--------------|------------|----------|
| | Dry Eyes | Blocked Nose | Dry Throat | Headache |
| Natural | 18(%) | 40(%) | 36(%) | 39(%) |
| Mechanical | 20 | 32 | 33 | 33 |
| Local induction/fan coil | 34 | 58 | 56 | 52 |
| Central induction/fan coil | 31 | 57 | 54 | 47 |
| All air | 31 | 45 | 46 | 43 |
| Whole group | 27 | 47 | 46 | 43 |

* Source: Data from Table 5 in Burge, et al., 1987.